EDITORIAL

Infectious Pericarditis

THE PERICARDIUM, an organ with limited and limiting functions, is a structure which man appears to be able to do without quite nicely, but which when injured can be the source of serious disease and life threatening pathophysiologic dysfunction. Like an actor with a limited repertoire, the pericardium when diseased can express its discomfiture in only several ways: by causing pain, by developing effusion or by fibrosing and constricting. The latter produces problems by restricting ventricular filling and interfering with the function of the enclosed heart or by a giant effusion compressing lung and interfering with pulmonary function.

From among all the diseases of the pericardium, in this issue of the JOURNAL, the Division of Infectious Diseases of the Harbor General Hospital focuses on the infectious causes of pericarditis in some detail. As usual, those problems which are still without definitive answers or which are still involved in controversy stimulate the most interest.

Idiopathic benign pericarditis remains the most common form of pericarditis in man. As is often the case with "idiopathic" disease, benign pericarditis is generally believed to be of viral cause.[1] Unfortunately, when the viral cause of idiopathic benign pericarditis is prospectively investigated, the virus continues to be elusive in most cases. For instance, Johnson and co-workers found evidence of a viral cause in five of 34 patients with acute benign pericarditis,[2] and Fowler in a prospective study of 25 cases of acute pericarditis found evidence of viral infection in only four.[1]

The inability to find the virus in these cases may be a manifestation of the limits in the techniques of viral identification and propagation that are presently available. Other explanations for our failure to grow virus from the patient's fluid or tissue, or to show active antibody response to viral antigen, are possible, however. It may be that viruses are only one of several causes of the clinical entity known as idiopathic benign pericarditis. Increasingly evident in experimental work is the fact that viral invasion can alter cells due to the continued presence of viral antigen or the development of antibodies to heart muscle such that persistent cellular dysfunction may occur long after the disappearance of viable virus. Such a mechanism may be responsible for the known tendency for idiopathic pericarditis as well as the other apparently noninfectious forms of pericarditis occurring after myocardial infarction and after pericardial injury and cardiac surgery to recur, often a number of times.[3]

Once the mechanism which results in recurrence is understood, we will be in a better position to prevent recrudescence and especially to treat the rare patient who has repeated episodes of disabling pericarditis and who presents an extremely difficult management problem. Occasionally in these patients it is necessary to employ analgesics, steroids, antimetabolite therapy[4] and finally pericardiectomy,[5] each associated with its own set of problems. The etiologic role of antiheart antibodies and autoimmune damage is as yet unclear in all forms of pericarditis. Engle and co-workers reported in a prospective, double-blind study, the close correlation of the presence of heart-reactive antibody in high titer in the serum of patients in whom the post-pericardiectomy syndrome developed after cardiac surgery.[6] The similarity of the clinical features of idiopathic pericarditis to those in the other forms of pericarditis—such as ones occurring after injury to the pericardium or after myocardial infarction—suggests a common underlying mechanism of injury. Such would be the case if each of the inciting injuries resulted in an autoimmune response which caused the clinical disease we recognize as pericarditis.

Tuberculous pericarditis remains a problem in diagnosis and management. The classical description of tuberculous pericarditis is that of a clinically insidious, chronic disease. It is less well

recognized that tuberculous pericarditis can initially manifest itself as typical acute pericarditis. The problem of the definitive identification of tuberculosis as the etiologic agent in pericarditis is of some importance since, unrecognized, the mortality approaches 80 to 90 percent with this disease.[7] Medical therapy alone appears to be less than totally satisfactory once the tuberculous nature of the pericarditis is recognized. Carson and associates reported that medical therapy in 62 consecutive patients with tuberculous pericarditis achieved satisfactory results in less than a third of the patients. Half of their patients required pericardiectomy for recurrent pericardial tamponade or constriction.[8]

With the increasingly smaller number of young people in this country for whom results are positive on tuberculin skin tests, purified protein derivative of tuberculin (PPD) has become more valuable in recognizing tuberculosis as a specific cause of pericarditis. A positive reaction on a skin test, however, simply tells us that the patient has had previous exposure to the tubercle bacillus, not that the pericarditis is caused by tuberculosis. At present, especially in young people with pericarditis, a positive reaction on a skin test is usually sufficient to start the patient on 12 to 18 months of antituberculous therapy. Even with the relative innocuousness of the drugs, though isoniazid and other antituberculous drugs are not without serious side effects, it would be preferable to establish the diagnosis positively before committing the patient to such a long and possibly difficult course of therapy.

Pericardiocentesis is helpful in definitely establishing the diagnosis but unfortunately at most only 50 percent of patients aspirated have positive cultures for tuberculosis.[9] If the usefulness of pleural biopsy in establishing the tuberculous cause in pleural effusion is analogous to pericarditis, the increased use of open pericardial biopsy should be considered more frequently, especially in younger patients in whom tuberculin skin tests give positive findings. If the biopsy specimen shows caseating granulomata or cultures Mycobacterium tuberculosis, the cause is established and the patient needs a full course of antituberculous therapy. If the biopsy specimen shows nonspecific pericarditis, the physician would be justified in withholding antituberculous therapy.

Because of the high incidence of constriction in tuberculous pericarditis, as mentioned, early diagnosis is most important. The role of steroids in preventing the development of constriction in tuberculous pericarditis is still unclear, and controlled studies to show the possible efficacy of this therapy are complicated by the relative rarity of the disease and therefore the difficulty in accumulating a large enough series in any one institution.

Once constrictive pericarditis is well established, the necessity for pericardiectomy is well recognized. Less well appreciated, especially by internists and cardiologists, are the problems confronting the surgeon who must find tissue planes and carry out an adequate pericardiectomy without injury to the coronary arteries and extensive damage to the underlying myocardium. Early identification of pericardial fluid organization or myocardial restriction would be beneficial in that early pericardiectomy could be done before extensive fibrosis involves the myocardium and obliterates cardiac surface structures. Early recognition of organizing pericardial fluid and early constriction could be recognized at the time of pericardial biopsy. The operative mortality from pericardiectomy, which at present can be as high as 29 percent,[10] would probably be reduced if the surgical procedure could be done early.

Of great interest is the changing nature of the infectious agents attacking the pericardium. To a large degree this changing flora is related to the improvement in our ability to treat infectious, malignant and immunologic diseases. With the introduction of antibiotics the incidence of pneumococcal percarditis, certainly among the commonest of organisms causing purulent pericarditis in the past, was notably reduced.[11] At present, with the manipulation of man's bacterial inhabitants by antibiotics and the interference of defense mechanisms by steroids and immunosuppressive agents, we are seeing the increasing incidence of infections, including purulent pericarditis, caused by saprophytes and previously nonpathogenic bacteria and fungi. A high index of suspicion allowing us to recognize purulent pericarditis is essential, for the penalty for overlooking the diagnosis is a mortality approaching 100 percent.[12]

Finally, although it is stated that constrictive pericarditis is unusual as a sequela of purulent pericarditis, it most certainly does occur and must not be overlooked. It has been especially noted in very young patients and can occur within a matter of weeks after the acute infection has been treated apparently adequately with antibiotics.[13] The frequent necessity for surgically draining the pericardium in purulent pericarditis must again be

emphasized and because of the possibility of constriction the patient must be followed carefully after the recognition of purulent pericarditis to detect the earliest signs of this complication.

It is obvious that there are still many areas both in the diagnosis and management of patients with pericardial disease where definitive answers are not presently available. It is interesting—and somewhat disappointing—that, as one looks back at the overview of pericardial disease written by Connolly and Burchell in 1961,[3] many of the same still unanswered questions are asked. As long as this is true, interest and research in the area of pericardial disease will continue to remain clinically important.

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Medical Accidents and Medical Accident Insurance

PERHAPS IT IS TIME to get back to the old idea that doctors are really trying to do their best in taking care of their patients and to recognize that, since doctors are human like other people, they may make mistakes even while they are trying to do their best. The mistakes may be in judgment or in something they do or do not do. In times past this was an accepted assumption and often there was little recourse for the patient who was the unfortunate victim of a doctor's mistake. This was wrong and there has been a great change since tort law became applied both extensively and effectively in the medical field. The extent of this redress adjudged by the courts in favor of patients has now become very great. It is becoming abundantly clear that the physician, the hospital or the insurance company can no longer pay the cost of these judgments and the expensive litigation that attends them, and there is beginning to be some question as to whether these costs should be passed on to the patient population or whether they should be spread over an even wider base.

But let us return to the idea that the doctor tries to do his best. Certainly there is no reason to believe that doctors want to do their worst or that they seek to damage or injure their patients. It seems that any such instance would or should be a criminal offense. Given that the intent is good, the limited numbers of mistakes and unfortunate outcomes which inevitably occur are more in the nature of accidents—unexpected events which occur and which none of the parties involved plan or expect to happen. If these incidents, tragic though they may be, are viewed as accidents much as injuries or diseases sustained while working for an employer are viewed, then the situation seems to clarify. It can then be admitted that medical accidents can and do happen and that no one need be particularly at fault even though errors in judgment or action may have been made. It can also be admitted that the patient or medical-accident victim is entitled to some acceptable compensation for his unlucky or unfortunate outcome. The costs could then be kept within the capability of the health care industry, which includes physicians, to pay. And fully as important, practicing physicians and hospitals could return to giving their whole attention to trying to do their best for the patient instead of